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Cholesterol crystal embolism: Diagnostic and treatment

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To the Editor: In his recent in-depth review of cholesterol crystal embolization syndrome (CCE), Meyrier[1] has delineated the pathogenesis, differential diagnoses, and therapeutic aspects of CCE. In addition to therapeutic measures of paramount importance such as restriction of further intravascular interventions, stop of anticoagulation, and treatment of renal insufficiency with dialysis, we would like to add the possibility of performing renal transplantation in selected CCE cases with end-stage renal disease and stable clinical course after diagnosis of CCE. We have previously reported a patient with CCE successfully undergoing renal transplantation.[2] According to the best of our knowledge, there are so far no other published cases of the renal transplantation after CCE-induced end-stage renal disease.

Briefly, a 63-year-old patient with a high load of atherosclerotic risk factors (heavy smoker, hypertension 160/80–180/100 mm Hg, severe hyperlipidemia (triglycerides up to 500 mg/dl; low-density lipoprotein cholesterol up to 240 mg/dl)) suffered from end-stage renal disease owing to cholesterol emboli after coronary angiography because of symptomatic coronary artery disease in October 1997. Within 1 week, the patient developed renal failure necessitating hemodialysis since December 1997. Smoking cessation, effective control of blood pressure ($<$130/80 mm Hg), and serum lipids (low-density lipoprotein cholesterol $<$100 mg/dl) was achieved and maintained until successful renal transplantation from a living related donor in 1998. Until his last follow-up in May 2006, kidney function has remained stable with a current serum creatinine level of 1.28 mg/dl, corresponding to a calculated creatinine clearance of 60 ml/min. Serum lipids have remained normalized with diet and pravastatin therapy (40 mg/day) with total cholesterol levels of about 187 mg/dl and low-density lipoprotein cholesterol levels of about 120 mg/dl as well as normotensive blood pressure levels have been achieved with losartan, doxazosin, nitrendipin, and nebivolol combined antihypertensive therapy, and the patient refrained from smoking.

In conclusion, secondary prevention of CCE, that is, rigid long-term control of the underlying atherosclerotic risk factors may enable a selected subgroup of patients with CCE to undergo successful renal transplantation with excellent long-term patient and graft survival.

1. Meyrier A. Cholesterol crystal embolism: diagnostic and treatment. *Kidney Int* 2006; **69**: 1308–1312.
2. Kammerl MC, Fischereder M, Zuelke C *et al.* Renal transplantation in a patient with end stage renal disease due to cholesterol embolism. *Transplantation* 2001; **71**: 149–151.

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Response to 'Cholesterol crystal embolism: Diagnostic and treatment'

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Kryvoshey *et al.*'s experience[1] with renal transplantation to treat end-stage renal disease following cholesterol crystal embolism to the kidney is quite interesting. That renal replacement was not complicated by relapse of cholesterol crystal embolism on the transplant confirms the interest of stabilizing atherosclerotic plaques with statins, as already shown by Scolari *et al.*[2] However, patients with massive cholesterol crystal embolism do not die from renal failure but from vital organ injury, such as mesenteric and pancreatic ischemia. From this standpoint, the writers of this letter confirm the essential role of preventing cholesterol crystal embolism in patients at risk, that is, lifelong smokers with lipid disorders and widespread atherosclerosis.

1. Kryvoshey D, Kammerl M, Hoffmann U *et al.* Cholesterol crystal embolism: Diagnosis and treatment. *Kidney Int* 2006 (in press).
2. Scolari F, Ravani P, Pola P *et al.* Predictors of renal and patient outcomes in atheroembolic renal disease: a prospective study. *J Am Soc Nephrol* 2003; **14**: 1584–1590.

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Cardiac troponins and chronic kidney disease

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To the Editor: We were interested in the recent paper by Kanderian and Francis[1] in which they review hypothetical mechanisms contributing to the increase in serum troponin concentrations observed in chronic kidney disease (CKD). The work of Diris *et al.*,[2] who demonstrated fragments of the cardiac troponin T (cTnT) molecule ranging in size from 8 to 25 kDa in hemodialysis patients, is discussed. It is suggested